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1. The Motor Area of the Human Cerebrum, Its Position and Subdivisions, with Some Discussion of the Surgery of This Area. Charles K. Mills and Charles H. Frazier.
2. The Occasional Clinical Resemblance Between Caries of the Vertebræ and Lumbothoracic Syringomyelia, and the Location within the Spinal Cord of the Fibers for the Sensations of Pain and Temperature. William G. Spiller.
3. A Case of Amyotrophic Lateral Sclerosis in Which the Symptoms Were Unilateral and Ascending. Charles S. Potts.
4. A Case of Tumor of the Left First and Second Frontal Convolutions with Motor Agraphia as Its Chief Localizing Symptom; Successful Removal of the Tumor. J W. McConnell.
5. A Study of the Contractures in Organic Nervous Diseases, and Their Treatment. T. H. Weisenburg.
6. The Difficulty of Diagnosticating Between Tabes and Cerebrospinal Syphilis, with a Report of Two Illustrative Cases. Carl D. Camp.

1. *The Motor Area of the Human Cerebrum.*—The history and literature of the subject is summarized, attention first being directed to the paper by Lamacq, published in 1897, on the cortical motor areas of the human brain, as determined by faradic excitation of the cerebral hemispheres of man. Lamacq included in his paper a series of unpublished cases furnished by Dr. W. W. Keen of Philadelphia. Most of the work done in this field has come from American neurologists and surgeons.

Since 1897, contributions on the subject referred to by the authors include recent articles by Krause, Rothmann and Brodman. Attention is directed to the observation on anthropoids of Grünbaum and Sherrington, who hold that the motor region is situated cephalad of the central fissure. The authors next recall in some detail, because of their historical value and the striking results obtained, three of the earliest series of observations made in Philadelphia in 1888, those of Keen, of Lloyd and Deaver and of Mills and Hearn. It is notable that in the case of Mills and Hearn the results obtained were all from applications made cephalad of the central fissure. Next a series of hitherto unpublished observations is given. In seven of these cases operation was performed by Dr. Frazier, the cases, as indicated in the summaries, having been in the care of Drs. Mills, Spiller or McConnell, or having been directly in the service of Dr. Frazier. Notes of the experimentation on three other hitherto unpublished cases are given, these having been furnished by Dr. W. W. Keen.

In order to get some data of value with regard to the position of the motor area with relation to the central fissure, an analysis is made of the results of faradic applications in twenty-five cases. The result of the analysis is given in tabular form as follows:

Number of cases	25
Number of applications	138
Responses in front of central fissure	62
Responses behind central fissure	20
Responses in uncertain position as to central fissure	14
No response in front of central fissure	14
No response behind central fissure	28

The data are altogether in favor of the view that the motor region is in front of the central fissure. Mills in 1888 wrote that the facts then obtained upheld the view that the motor zone, in man at least, is much more extensive in front of than behind the fissure of Rolando. The exact limitations and main subdivisions of the motor zone, according to the views of the writers, are then given. This zone is placed entirely cephalad of the central fissure and largely in the precentral convolution, with a forward projection which includes portions of the second and third frontal gyres. The motor zone and its subdivisions in the human brain are represented in a new scheme or diagram. The subdivisions of the areas for the face, the eyes and the head, the arm, the trunk and the leg are then discussed in detail. Experiments on the cadaver were performed to corroborate the accuracy of the methods employed, not only for exposing the central fissure and the lesion related to it, but also for the selection of the points to be faradized. The exact method of using the current is given. In the observations of the authors, the method of unipolar faradization was employed, in most of the previously published cases, Keen's bipolar electrode having been used. Grünbaum, Sherrington and Krause, in their observations on the anthropoids, resorted to unipolar faradization, and the authors discussed its value, and also the necessity of an exact and careful technique. The paper closes with a discussion of the subject of craniometric lines and craniocerebral relations. It contains five illustrations, namely: (1) A scheme of the motor zone and its subdivisions; (2) the Anderson-Makins lines and the usual osteoplastic flap for exposing the motor region; (3) bony and dural flaps reflected so as to show the convolutions of the motor area (4) reflected osteoplastic flap in cadaver operation, showing the places of insertion of pins at points of selection for precentral and postcentral faradization; (5) drawing of the brain after calvarium and membranes were removed, showing the position of osteoplastic opening with reference to the fissures and gyres of the motor region. (Author's abstract.)

2. *Lumbothoracic Syringomyelia and Caries of the Vertebræ.*—Attention is called to the infrequency of lumbar or sacral syringomyelia, and reference is made to the few cases in the literature. The following case observed by the author is reported: A woman, aged at present thirty-six years, has symptoms confined to the lower limbs, if inequality of the pupils is excluded. In the right lower limb pain and temperature sensations are much diminished; while tactile sensation is prompt, the patellar tendon reflex and Achilles tendon reflex are lost; the limb is flaccid, and its movements in walking seem to be a little ataxic, while voluntary power is about normal. In the left lower limb all forms of sensation are normal, weakness is distinct, the patellar reflex is exaggerated, and patellar clonus and ankle clonus are present; the limb is a little spastic, and its toes are dragged a little when the patient walks. Babinski's reflex is present on each side. It is a Brown-Séquard paralysis (weakness in the left lower limb, dissociation of sensation in the right lower limb), with spasticity and exaggerated tendon reflexes in the left lower limb, and flaccidity and loss of tendon reflexes in the right lower limb.

A lesion in the left half of the spinal cord in the thoracic region and above the lumbar region would explain the weakness of the left lower limb and the exaggeration of the tendon reflexes and the presence of the Babinski reflex in the left lower limb; also the dissociation of sensation (reservation of tactile sensation, with more impairment of temperature and pain sensations) in the right lower limb, because the sensory fibers from the right lower limb cross soon after entering the cord to the left side. We must assume that the crossed pyramidal tract and other parts of the lateral column, especially Gowers's tract on the left side, are affected, but the gray matter of the anterior and posterior horns of this side cannot be diseased in the lumbar region, or there would be atrophy and loss of tendon reflexes in the left lower limb. To explain the loss of tendon reflexes and the flaccidity in the right lower limb without atrophy, we must assume that there is a lesion of the right posterior horn of the lumbar region without involvement of the right anterior horn, as no atrophy has occurred, even after two years, and we should expect atrophy were the anterior horn cells diseased. The lesion may affect slightly the right crossed pyramidal tract, because of the Babinski reflex and possibly slight weakness on the right side, but it cannot greatly impair this tract, as the weakness of in the left lower limb. The lesion must, therefore, involve much of the right lateral column, as sensations of pain and temperature are normal in th eleft lower limb. The lesion must, therefore, involve much of the left half of the cord in the thoracic region, but not extend into the left lumbar region very far, and must implicate the right posterior horn in the lumbar region; it must also be a slowly developing lesion, and nothing suits these conditions so well as syringomyelia. A second case is as follows: A man, aged twenty-three years, had resistance to passive movements slightly diminished in the lower limbs. Sensation for touch was normal or nearly normal in the lower limbs, but sensation for pain was abolished or almost abolished in these parts. Temperature sensation was almost lost in the lower limbs below the knees, and much impaired in the thighs and lower part of the trunk. Walking was difficult because of the deformity of the knees. He had a slight kyphosis at about the tenth thoracic vertebra. The upper limbs and face were not affected.

After a fall backward down a flight of steps he became completely paralyzed in the lower limbs. Sensation for touch was then lost in the right lower limb for a time, but was preserved in the left lower limb. Sensations for pain and temperature were lost in the right leg and foot and in the left foot, but were preserved elsewhere, although it is not stated that they were normal elsewhere. The patellar reflexes were much exaggerated, and Babinski's sign was present.

Still later sensation for touch was found preserved everywhere in the lower limbs. Sensation for pain was irregularly present in the lower limbs, the response to pain stimulation being very uncertain, and sensation for temperature was also much impaired in the lower limbs.

Tuberculous meningitis and caries of the vertebræ were found, and a small tubercle was present in the right lateral column at the extreme lower end of the thoracic cord, involving the area of Gowers's tract. About one-half to one inch higher another small tubercle was found involving the left tract of Gowers. Although there was some myelitis, it was very evident from the symptoms that it had chiefly developed after the fall down the flight of steps, as at the time the patient first came under the author's observation weakness in the lower limb was slight. (See this Journal, 1905, p. 318.) It is important to note that in this case the lesions of long duration were evidently the tubercles in Gowers's tracts, and they best explain the dissociation of sensation of the syringomyelic type; also that when the patient first came under observation there was probably little diffusion of the lesions because of the very slight motor involvement. (Author's abstract.)

3. *Amyotrophic Lateral Sclerosis.*—After reference to ten similar cases collected by Spiller ("Primary Degeneration of the Pyramidal Tracts," etc., *Jni. of Penna. Med. Bulletin*, vol. xvii, p. 390), the history of the case follows: A man, carpenter by occupation, who had worked in a lead works for the past eighteen months. Nothing else of note in the previous history. Six months before coming under observation he first noticed weakness of the left leg. Several weeks after this, weakness was noted in the left arm, and still later bulbar symptoms appeared. Examination revealed weakness of the muscles about the angles of the mouth on both sides; weakness and atrophy of the tongue; difficulty in swallowing; a slurring and indistinct speech, with impairment of motion of the right vocal cord. There was marked weakness of the left arm and leg; a much

less degree on the right side; marked atrophy of intrinsic hand muscles with claw hand deformity on the left, slight atrophy of the same on the right. The left leg was very spastic and the foot was dragged when he walked. The Babinski reflex was present on both sides, and all of the tendon jerks (arms and legs) were increased. Fibrillary tremors were observed. There was diminished faradic contractility in the atrophied muscles, and he was very emotional. This patient[1] was exhibited to the Philadelphia Neurological Society. (Author's abstract.)

4. *Tumor of the Left First and Second Frontal Convolutions, with Motor Agraphia.*—The author of this paper first speaks briefly of the difference of opinion with reference to the existence of a separate motor agrapnic center, and then presents the details of an interesting case. A right-handed man, twenty-eight years old, without syphilitic history and without any preceding history of importance except that he had had a fall when eight years old, striking on the occiput, had a convulsion for the first time in February, 1899. One year later he had a second seizure, six months later a third, a fourth in December, 1900, and in June, 1901, a series of four attacks in three days. After this he had convulsive attacks somewhat frequently, these, from the description of his wife, appearing to be general and attended with unconsciousness. In May, 1904, he began to have frequent local spasmodic attacks without unconsciousness, these having been as numerous as twelve in two hours. In these attacks the right side of the face was affected, including movements of both eyelids and sometimes rotation of the head to the right. After the attacks he was confused and his speech was thick. He was admitted to the Hospital of the University of Pennsylvania, May 18, 1904. Examination shortly after admission showed paresis on the right side of the face, including the levator palpebræ and palpebral orbicular muscles. Sensation seemed to be less acute on the right side of the face, as evidenced by the less amount of wincing of the patient on examination. Speech was thick, but by close attention what he said could be made out. The speech defect appeared to be due to paresis of the muscles of articulation, enunciation and phonation. He had no difficulty in reading print, writing or numbers, or in recognizing and correctly naming objects. He could not write either spontaneously or from dictation, although he held his pencil correctly; but writing from copy was practically perfect. A letter written two years before coming under observation showed omissions, faulty spelling and formation of words and letters in one part, which words were elsewhere spelled correctly or the letters properly formed. Specimens of later date show an exaggeration of these conditions up to the point of complete inability to write, as evidenced in the specimen of May 19, 1904. His memory and powers of attention had considerably failed. Examination showed no optic neuritis. Operation was performed by Dr. Charles H. Frazier, May 21, 1904. The anterior margin of the opening was two inches in front of the central fissure. The opening was subsequently enlarged in a forward and upward direction. A tumor was removed which was apparently situated so as to lie across the foot of the second fontal convolution, encroaching somewhat on the lower half of the first, slightly upon the upper posterior portion of the third and the anterior edge of the precentral convolution. After the operation, paresis of the right side of the face and to some extent of the right hand were present. Speech was immediately better, but the agraphia persisted. Speciments of his attempts at writing both before and after the operation were given. Gradually after the operation his writing improved so that by June, 1904, he wrote a letter of more than a page, of good construction and almost perfect spelling. A specimen of his writing at this time is also given. The history of the subject of motor agraphia is briefly reviewed, including a reference to the

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[1] JOUR. NERV. AND MENT. DIS., 1900, p. 556.

cases of Charcot and Dutil, Eskridge and Gordinier. The case is regarded by the author as additional evidence of the existence of a motor graphic center at the foot of the second frontal convolution. It has other interesting features, including a reference to some observations on cortical faradization. (Author's abstract.)

5. *Contractures in Organic Nervous Diseases.*—The results were based upon a study of about 500 cases of organic nervous disease. Of this number there were 184 cases of adult hemiplegia and 32 infantile hemiplegics. Contractures may be active or passive. By passive contractures is meant a condition due to disease of the part as joint disturbances; they are mostly not due to disease of the central nervous system, in contradistinction to active contractures which always have such an etiology. The so-called early contracture of hemiplegia are not always due to a ventricular lesion. More probably the same cause which partially interrupts the continuity of the motor fibers may cause an irritation. Of course it is not to be expected that early contractures or clonic spasms could appear in a total destruction of the motor fibers. The extra-pyramidal tracts are so little developed in man that they hardly could assume an immediate function. It seems probable that if early contractures or spasms appear, the motor tracts are not totally cut off and therefore we may give a better prognosis. This is an important point, and has not before been called attention to. The variable nature of the early contractures can be explained by the fact that the irritation is abortive and is soon exhausted. Besides most of the motor fibers have been cut off and an impulse would therefore be insufficiently carried and would be diffused to irregular groups of muscles. The late contractures of hemiplegia are fully discussed. No instance was found of what could be considered a bilateral contracture due to a unilateral cerebral lesion. In five such cases considered clinically, necropsy denoted bilateral lesions. Various authors consider this condition rare. In the literature, Spiller's case is the best and was carefully studied. This was due to a congenital unilateral hydrocephalus, and the bilateral contractures were probably due to an insufficient development of the motor fibers. The conclusion can be drawn that a unilateral cerebral lesion cannot cause a bilateral contracture in an adult, and that bilateral contractures are nearly always due to bilateral cerebral lesions. The form of the contractures due to bilateral cerebral lesions seems to vary according to the nature and the time of life the injury was sustained; thus the clinical picture and the contractures differ in the cases in which the lesions occur either before or at birth, and in those in which they occur in the adult. Mostly, the contractures in such diseases are in flexion both in the upper and the lower limbs. The causes that lead to contractures in cerebral disease are next considered. The different hypotheses are discussed and the following explanation offered. If we compare the position of the contractured limbs in a hemiplegia it will be found to be identical with the position most commonly assumed when at rest. Normally there is always an excess of strength and tonicity in certain muscle groups over their antagonists, and in the return of power after an apoplectic attack this physiological relation is maintained. The best explanation for the return of function in the paralyzed limbs after a destruction of the pyramidal fibers is probably found in the influence exerted by the extra pyramidal tracts. The nature of the bilateral contractures in bilateral lesions can be explained through mechanical grounds. In a unilateral involvement of pyramidal tracts, as in a hemiplegia, the contracture in the upper limb is in flexion, in the lower limb in extension. But if both pyramidal tracts are involved all of the contractures are in flexion. There is no organic reason for this. Its occurrence can be best explained by the fact that such a patient will naturally flex his limbs because he is more comfortable in this position, especially if there be pain. Again, if the disease process is slow as in some spinal diseases the patient will make these contractures permanent.

Two cases of hemihypertonia post apoplectica are recorded. The contractures in spinal cord diseases in peripheral nerve involvements and in muscle diseases are considered. Attention is called to the resemblance of the contractures found in paralysis agitans and arthritis deformans, substantiating Spiller's observation. The treatment of contractures is next considered. (Author's Abstract.)

6. *Tabes and Cerebro-Spinal Syphilis.* Two cases of cerebro-spinal syphilis are reported that, both climically and pathologically, bore a very close resemblance to cases of tabes dorsalis; the degeneration in the spinal cord being practically confined to the posterior columns. It is pointed out that this form of degeneration would occur if the posterior roots had been compressed to the point of destruction by a syphilitic meningitis, also on account of the blood supply of the spinal cord the posterior columns would suffer severely in any process which, by affecting the meninges would interfere with the circulation in the centripetal system of arteries. In as much as the diagnosis between tabes dorsalis and these cases of syphilitic pseudo-tabes is often impossible it would be a wise measure to treat all cases in which tabes is suspected with a thorough course of anti-syphilitic remedies. (Author's Abstract.)

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(Band 24—Heft 5-6.)

11. The Abdominal Symptom-Complex in Disease of the Lower Dorsal Cord, Its Roots and Nerves. By Oppenheim.
12. Subcortical Alexia with Agraphia and Apraxia. By Strohmayer.
13. Contribution to the Casuistry of Transcortical Motor Aphasia. By Strohmayer.
14. Remarks Upon Stuttering. By Maas.
15. The Behavior of the Axis Cylinders in Multiple Sclerosis. By Bartels.
16. Contribution to the Casuistry of Acute Hemorrhagic Encephalitis. By Rosenfeld.
17. Hemihypertrophia Facialis Progressiva. By Hoffmann.
18. Contribution to the Etiology of Tabes, with Especial Consideration of the Relation Between Injury and Tabes. By Schittenhelm.
19. Micropsia and Macropsia. By Veraguth.
20. Certain Forms of Amusia, Illustrated by Examples. By Würtzen.
21. Two Cases of Hysteria Characterized by Transitory Absence of the Patellar Reflex. By Nonne.

11. *Disease of Lower Dorsal Cord.*—Oppenheim discusses at considerable length the so-called abdominal symptom-complex. This concerns chiefly the behavior of the abdomen, and particularly the abdominal reflex in various diseases of the muscles, nerves or spinal cord which cause irregularities in the movements of the abdominal wall. His first patient had almost complete paralysis of the muscles on the left side of the abdomen, with the reactions of degeneration, and loss of reflexes on the left side. When the patient contracted the abdominal muscles the umbilicus deviated to the right. The second case, a man of fifty, had paresis of the abdominal muscles of the right side, with deviation of the umbilicus to the left, and a weaker abdominal reflex. The third case, a man of thirty-eight, had paralysis of the muscles of the left half of the abdomen, and symptoms similar to those of the first case. The diagnosis in all the cases was that of neuritis. Somewhat similar symptoms are freqeuntly caused by tumors of the spinal cord. Oppenheim reports four cases of this condition. Two of spondylitis tuberculosa, and one of doubtful spinal syphilis. There was also a doubtful case of multiple sclerosis giving the abdominal symptom complex, one of acute anteriopoliomyelitis occurring in a man of twenty-eight, and one of progressive muscular dystrophy. Oppenheim believes that the paralysis or paresis of the abdominal muscles is the most valuable element in this system group. The abdominal reflex